Towards adipose tissue-derived stromal cells-based therapy for diabetic retinopathy

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Propositions:

1. Adipose tissue derived stromal cells (ASC) support newly formed vascular networks by endothelial cells (EC) in vitro and in vivo through maintenance of the vascular architecture (this thesis).

2. Administered ASC to the murine model of diabetic retinopathy augment and stabilize retinal angiogenesis and co-localize with capillaries at a pericyte-specific position (this thesis).

3. The ROS-induced mitochondrial dysfunction, hyperglycemia-induced apoptosis, and bioenergetics changes only partially influence the pericytic ability of ASC (this thesis).

4. Despite of the diminished level of glycolysis, ASC under HG showed the same basal respiration as under NG which proves that ASC resist the HG condition by changing the metabolic limit deprived of altering the proliferation rate (this thesis).

5. Conditioned medium of ASC alleviates high glucose-induced oxidative stress and its subsequent upregulated downstream targets in an NF-κB dependent fashion in bovine retinal endothelial cells (this thesis).

6. The manner of high glucose preconditioning of adipose tissue-derived stromal cells dictates their immuno-regulatory properties (this thesis).

7. The antioxidant capacity of ASC-Cme downmodulates upregulated pro-inflammatory genes in high glucose-challenged BREC (this thesis).

8. The new pharmacologic compound, (6-hydroxyl-2,5,7,8-tetramethylchroman-2-yl)(4-(2-hydroxyethyl)piperazin-1-yl) methanone (SUL-109) shields ASC during cell preservation from hypothermic cell death without influencing their multi-potency capacity and proliferation (this thesis).